09/732,360.

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Search Results -

Terms	Documents
(metabolic pathway and GA binding protein)and (microcompetition) and diseases	0

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Derwent World Patents Index
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Refine Search: (metabolic pathway and GA binding protein)and (microcompetition) and diseases Clear

Search History

Today's Date: 1/24/2002

DB Name	Query	Hit Count	Set Name
USPT	(metabolic pathway and GA binding protein)and (microcompetition) and diseases	0	L3
USPT	(metabolic pathway and GA binding protein)and diseases	68000	L2
USPT	(metabolic pathway and GA binding protein)	224332	L1

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File copy (STN Medline)

```
=> s metabolic pathway
        157412 METABOLIC
             9 METABOLICS
        157416 METABOLIC
                 (METABOLIC OR METABOLICS)
        128587 PATHWAY
        131568 PATHWAYS
        235501 PATHWAY
                 (PATHWAY OR PATHWAYS)
L1        6310 METABOLIC PATHWAY
                 (METABOLIC(W)PATHWAY)

=> s GA binding protein
          6061 GA
        114036 GAS
        119865 GA
                 (GA OR GAS)
        564198 BINDING
          1137 BINDINGS
        564449 BINDING
                 (BINDING OR BINDINGS)
       1030658 PROTEIN
        869837 PROTEINS
       1341427 PROTEIN
                 (PROTEIN OR PROTEINS)
L2          65 GA BINDING PROTEIN
                 (GA(W)BINDING(W)PROTEIN)

=> s l1 and l2
L3           0 L1 AND L2

=> s l2 and risk factor
        475338 RISK
         51648 RISKS
        497162 RISK
                 (RISK OR RISKS)
        511281 FACTOR
       1429669 FACTORS
       1762656 FACTOR
                 (FACTOR OR FACTORS)
        225852 RISK FACTOR
                 (RISK(W)FACTOR)
L4           0 L2 AND RISK FACTOR

=> s cellular microcompettion
        244936 CELLULAR
             2 CELLULARS
        244937 CELLULAR
                 (CELLULAR OR CELLULARS)
             0 MICROCOMPETTION
L5           0 CELLULAR MICROCOMPETTION
                 (CELLULAR(W)MICROCOMPETTION)

=>
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File copy

```
 s GABP metabolic pathways
           125 GABP
             1 GABPS
           125 GABP
                 (GABP OR GABPS)
        156905 METABOLIC
             9 METABOLICS
        156909 METABOLIC
                 (METABOLIC OR METABOLICS)
        130947 PATHWAYS
L1           0 GABP METABOLIC PATHWAYS
                 (GABP(W)METABOLIC(W)PATHWAYS)

=> s GABP
           125 GABP
             1 GABPS
L2         125 GABP
                 (GABP OR GABPS)

=> s disruption and l2
         32869 DISRUPTION
          2482 DISRUPTIONS
         34790 DISRUPTION
                 (DISRUPTION OR DISRUPTIONS)
L3           3 DISRUPTION AND L2

=> d l3 ibib abs total

L3   ANSWER 1 OF 3     MEDLINE
ACCESSION NUMBER:    1998343943     MEDLINE
DOCUMENT NUMBER:     98343943   PubMed ID: 9677314
TITLE:               4-Aminobutyrate (GABA) transporters from the
                     amine-polyamine-choline superfamily: substrate specificity
                     and ligand recognition profile of the 4-aminobutyrate
                     permease from Bacillus subtilis.
AUTHOR:              Brechtel C E; King S C
CORPORATE SOURCE:    Department of Physiology and Biophysics, University of
                     Texas Medical Branch, Galveston, Texas 77555-0641, USA.
CONTRACT NUMBER:     T32-ES07254 (NIEHS)
SOURCE:              BIOCHEMICAL JOURNAL, (1998 Aug 1) 333 ( Pt 3) 565-71.
                     Journal code: 9YO; 2984726R. ISSN: 0264-6021.
PUB. COUNTRY:        ENGLAND: United Kingdom
                     Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:            English
FILE SEGMENT:        Priority Journals
ENTRY MONTH:         199809
ENTRY DATE:          Entered STN: 19981006
                     Last Updated on STN: 19981006
                     Entered Medline: 19980923
AB   A previous study [Ferson, Wray and Fisher (1996) Mol. Microbiol. 22,
     693-701] has shown that transposon-mediated disruption of a
     protein 47% identical to the Escherichia coli GABA (4-aminobutyrate)
     transporter abolishes the ability of nitrogen-limited culture conditions
     to induce expression of a GABA transport activity in Bacillus subtilis.
     Here it is demonstrated directly that the B. subtilis GABA permease (
     gabP) gene can complement the transport defect in the gabP
     -negative E. coli strain. Unexpectedly, the ligand-recognition profile of
     the B. subtilis GabP was found to differ substantially from that
     of the highly homologous E. coli GabP. Unlike the E. coli
     GabP, the B. subtilis GabP: (i) exhibits approx. equal
     preference for the 3-carbon (beta-alanine, Km=9.6 microM) and the 4-carbon
     (GABA, Km=37 microM) amino acids, and (ii) resists inhibition by bulky,
```

conformationally constrained compounds (e.g. nipecotic acid, guvacine), which are active against GABA transporters from brain. The present study shows additionally that the B. subtilis **GabP** can translocate several open-chain GABA analogues (3-aminobutyrate, 3-aminopropanoate, cis-4-aminobutenoate) across the membrane via counterflow against [3H]GABA. Thus, consistent with the idea that the ligand-recognition domain of the B. subtilis **GabP** is less spacious than that of the close homologue from E. coli, the former exhibits more stringent requirements than the latter for substrate recognition and translocation. These distinct functional characteristics of the E. coli and B. subtilis GABA transporters provide a basis by which to identify ligand-recognition domains within the amine-polyamine-choline transporter superfamily.

L3 ANSWER 2 OF 3 MEDLINE
ACCESSION NUMBER: 97439460 MEDLINE
DOCUMENT NUMBER: 97439460 PubMed ID: 9295016
TITLE: PU.1 negatively regulates the CD11c integrin gene promoter through recognition of the major transcriptional start site.
AUTHOR: Lopez-Rodriguez C; Corbi A L
CORPORATE SOURCE: Hospital de la Princesa, Madrid, Spain.
SOURCE: EUROPEAN JOURNAL OF IMMUNOLOGY, (1997 Aug) 27 (8) 1843-7. Journal code: EN5; 1273201. ISSN: 0014-2980.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199709
ENTRY DATE: Entered STN: 19971013
Last Updated on STN: 19971013
Entered Medline: 19970930

AB CD11c integrin expression is restricted to myeloid cells and activated B lymphocytes, mainly through the collaborative action of Sp1 and members of the AP-1 and C/EBP transcription factor families on the proximal region of the CD11c gene promoter. While analyzing the role of an initiator-like sequence at the major transcriptional start site, an inverted consensus GGAA Ets binding site was identified as a negative regulatory element whose **disruption** increases the activity of the CD11c promoter. The GGAA element was specifically recognized by PU.1 in THP-1 monocytic cells and by PU.1 and **GABP**-related proteins in U937 promonocytic cells. Mutational analysis indicated that PU.1 recognition depends not only on the GGAA consensus element but also on flanking sequences. The functional relevance of PU.1 binding was assayed in transactivation experiments in HeLa cells, where PU.1 co-expression led to a significant decrease in the activity of the CD11c promoter, demonstrating that PU.1 inhibits the activity of the CD11c promoter through a PU.1 binding site located at the major transcriptional start site (PU1-5). The inhibitory action of PU.1 on CD11c is in contrast with its positive regulatory effect on the CD11b and CD18 integrin gene promoters, which might contribute to the differentially regulated expression of CD11b/CD18 and CD11c/CD18 during monocyte extravasation and terminal maturation. In addition, since PU.1 transcriptional activity correlates with macrophage proliferation, PU.1 might modulate CD11c gene transcription according to the proliferative state of the cell.

L3 ANSWER 3 OF 3 MEDLINE
ACCESSION NUMBER: 96007509 MEDLINE
DOCUMENT NUMBER: 96007509 PubMed ID: 7559529
TITLE: **GABP** and PU.1 compete for binding, yet cooperate to increase CD18 (beta 2 leukocyte integrin) transcription.
AUTHOR: Rosmarin A G; Caprio D G; Kirsch D G; Handa H; Simkevich C P

CORPORATE SOURCE: Brown University School of Medicine, Division of Hematology, Miriam Hospital, Providence, Rhode Island 02906, USA.
CONTRACT NUMBER: R29 DK 44728 (NIDDK)
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1995 Oct 6) 270 (40) 23627-33.
Journal code: HIV; 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199511
ENTRY DATE: Entered STN: 19951227
Last Updated on STN: 19951227
Entered Medline: 19951114

AB CD18 (beta 2 leukocyte integrin) is a leukocyte-specific adhesion molecule that plays a crucial role in immune and inflammatory responses. A 79-nucleotide fragment of the CD18 promoter is sufficient to direct myeloid transcription. The CD18 promoter is bound by the B lymphocyte- and myeloid-restricted ets factor, PU.1, and **disruption** of the PU.1-binding sites significantly reduces promoter activity. However, PU.1 alone cannot fully account for the leukocyte-specific and myeloid-inducible transcription of CD18. We identified a ubiquitously expressed nuclear protein complex of extremely low electrophoretic mobility that also binds to this region of the CD18 promoter. This binding complex is a heterotetramer composed of **GABP** alpha, and ets factor, and **GABP** beta, a subunit with homology to Drosophila Notch. **GABP** alpha competes with the lineage restricted factor, PU.1, for the same critical CD18 ets sites. The CD18 promoter is activated in myeloid cells by transfection with both **GABP** alpha and **GABP** beta together, but not by either factor alone. Transfection of non-hematopoietic cells with the two **GABP** subunits together with PU.1 is sufficient to activate CD18 transcription in otherwise non-permissive cells. Thus, **GABP** and PU.1 compete for the same binding sites but cooperate to activate CD18 transcription.

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                                                   ENTRY    SESSION
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=> s l3
L4          11 L3

=>
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=> dup rem l4
PROCESSING COMPLETED FOR L4
L5             3 DUP REM L4 (8 DUPLICATES REMOVED)

=> d l5 ibibabs
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IALL -------- ALL, indented with text labels
IBIB -------- BIB, indented with text labels
IIND -------- IND, indented with text labels
IND --------- NCL, CC, BC, IT (CT, ST), CO, NA, NP, GT, ORGN, RN

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HITIND ------ IND
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specified Accession Number.
ENTER DISPLAY FORMAT (BIB):
<------User Break------>

ENTER DISPLAY FORMAT (BIB):
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```
 s human GABP
       7503616 HUMAN
         97087 HUMANS
       7522518 HUMAN
                 (HUMAN OR HUMANS)
           125 GABP
             1 GABPS
           125 GABP
                 (GABP OR GABPS)
L1           1 HUMAN GABP
                 (HUMAN(W)GABP)

=> s cellular microcompetition
        244275 CELLULAR
             2 CELLULARS
        244276 CELLULAR
                 (CELLULAR OR CELLULARS)
             0 MICROCOMPETITION
L2           0 CELLULAR MICROCOMPETITION
                 (CELLULAR(W)MICROCOMPETITION)

=> s microcompetition and human diseases
             0 MICROCOMPETITION
       7503616 HUMAN
         97087 HUMANS
       7522518 HUMAN
                 (HUMAN OR HUMANS)
       1305724 DISEASES
          2466 HUMAN DISEASES
                 (HUMAN(W)DISEASES)
L3           0 MICROCOMPETITION AND HUMAN DISEASES

=> s l1 and diseases
       1305724 DISEASES
L4           0 L1 AND DISEASES

=>
```

```
=> s GABP
           125 GABP
             1 GABPS
L5         125 GABP
                 (GABP OR GABPS)

=> s diseases
L6     1305724 DISEASES

=> s l5 and l6
L7           1 L5 AND L6

=> d l7 ibib abs

L7   ANSWER 1 OF 1     MEDLINE
ACCESSION NUMBER:    2000087178     MEDLINE
DOCUMENT NUMBER:     20087178   PubMed ID: 10618716
TITLE:               Ets transcription factors cooperate with Sp1 to activate
                     the human tenascin-C promoter.
AUTHOR:              Shirasaki F; Makhluf H A; LeRoy C; Watson D K; Trojanowska
                     M
CORPORATE SOURCE:    Department of Medicine, Division of Rheumatology, Hollings
                     Cancer Center, Medical University of South Carolina,
                     Charleston, South Carolina, SC 29425-2229, USA.
CONTRACT NUMBER:     AR42334 (NIAMS)
SOURCE:              ONCOGENE, (1999 Dec 16) 18 (54) 7755-64.
                     Journal code: ONC; 8711562. ISSN: 0950-9232.
PUB. COUNTRY:        ENGLAND: United Kingdom
                     Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:            English
FILE SEGMENT:        Priority Journals
ENTRY MONTH:         200002
ENTRY DATE:          Entered STN: 20000218
                     Last Updated on STN: 20000218
                     Entered Medline: 20000204
AB   Tenascin-C (TN-C), an extracellular matrix glycoprotein is expressed
     during embryonic development, but is present only at low levels in normal
     adult tissues. TN-C is re-expressed during wound healing, fibrotic
     diseases and in cancer. To better understand the mechanisms that
     control TN-C gene expression, we examined the regulation of the human TN-C
     promoter in human fibroblasts. We demonstrate that a short segment of the
     TN-C promoter between bp -133 and -27 contains three evolutionarily
     conserved Ets binding sites (EBS). These three EBSs bind in vitro
     expressed Fli1 protein and mediate transactivation of the TN-C gene by
     Fli1. Furthermore, two proximal EBSs contribute significantly to basal
     activity of the TN-C promoter. GABP, which is present in human
     fibroblast nuclear extracts, interacts with the two proximal EBSs. In
     addition, several Sp1 and Sp3 binding sites have been located in close
     proximity to the EBSs within this promoter region. The studies performed
     in Drosophila cells demonstrate that either Fli1 or GABPalpha+beta1
     functionally interact with Sp1 resulting in a synergistic stimulation of
     the TN-C promoter activity. In conclusion, this study shows for the first
     time that the TN-C gene is regulated by Ets proteins, which together with
     Sp1 act as potent activators of TN-C expression.

=>
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1 ANSWER 1 OF 1 MEDLINE
ACCESSION NUMBER: 97169219 MEDLINE
DOCUMENT NUMBER: 97169219 PubMed ID: 9016666
TITLE: Functional domains of transcription factor hGABP beta1/E4TF1-53 required for nuclear localization and transcription activation.
AUTHOR: Sawa C; Goto M; Suzuki F; Watanabe H; Sawada J; Handa H
CORPORATE SOURCE: Faculty of Bioscience and Biotechnology, Tokyo Institute of Technology, Midori-ku, Yokohama, Japan.
SOURCE: NUCLEIC ACIDS RESEARCH, (1996 Dec 15) 24 (24) 4954-61. Journal code: O8L; 0411011. ISSN: 0305-1048.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199703
ENTRY DATE: Entered STN: 19970321
Last Updated on STN: 19970321
Entered Medline: 19970311

AB Transcription factor E4TF1 is the human homolog of GABP and has been renamed hGABP (**human GABP**). hGABP is composed of two types of subunits; hGABP beta1/E4TF1-53 and the ets-related protein hGABP alpha/E4TF1-60. Both bind together to form an (alpha)2(beta1)2 heterotetrameric complex on DNA and activate transcription at specific promoters in vitro. Tetramer formation depends on two regions of hGABP beta1; the N-terminal region containing the Notch/ankyrin-type repeats is necessary for binding to hGABP alpha and the C-terminal region is necessary for homodimerization. In this report, we constructed various deletion mutants of hGABP beta1 in order to delimit the functional regions required for nuclear localization and transcription activity. We found that hGABP beta1 localization in the nucleus is dependent on a region located between amino acids 243 and 330 and that the presence of hGABP beta1 influences the efficiency of hGABP alpha transport into the nucleus. Next, we demonstrated that the hGABP complex composed of alpha and beta1 subunits activates transcription from the adenovirus early 4 promoter in vivo. This transcription activation needs the C-terminal region of hGABP beta1 and is consistent with results obtained with the in vitro assay. Furthermore, site-directed mutagenesis analysis of the C-terminal region reveals that the alpha-helix structure and the leucine residues are important for formation of a heterotetrameric complex with hGABP alpha in vitro and for transcription activation in vivo. These results suggest that hGABP beta1 stimulates transcription as part of a heterotetrameric complex with hGABP alpha in vivo.

=>